Section of Neurology

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DISCUSSION ON DIFFERENTIAL DIAGNOSIS AND TREATMENT OF POST-CONTUSIONAL STATES

Air Commodore C. P. Symonds: I shall assume that we are discussing the individual who has recovered from the acute stage of his head injury and is now in the chronic, or ambulant stage.

The problem of differential diagnosis and treatment in such cases may be divided into two. First there is the case in which the neurologist has seen the patient in the acute stage and has been able to observe subsequent progress. This is relatively simple. The second type of case is that in which the patient is first seen in the post-contusional or ambulant stage. (Ambulant, of course, must not be taken too literally. In many of these patients there are associated injuries which necessitate their being still in bed.) These cases are not only the most difficult, they are also the most common. Patients with head injuries are usually admitted to the nearest hospital and it is generally some time before the neurologist sees them. What, at this stage, is the general nature of the problem? It is that of a patient with a variety of subjective complaints, little in the way of abnormal physical signs, and, regrettably often, a quite inadequate record of the early stages of his illness.

Accurate diagnosis and correct treatment in such a case are of the utmost practical importance and the problem is one which demands a considerable expenditure of time if we are to arrive at any satisfactory solution. Here at once is a practical difficulty. Cases of this type should preferably be seen by appointment, or admitted to hospital for observation.

Reconstruction of the injury.—Our problem begins with the reconstruction of the story of the injury, often with scant information apart from what the patient can tell us. Fortunately the retrograde and post-traumatic amnesias can be estimated with rough accuracy at this stage, but it is important, if we are going to use these estimates, that we should standardize our end-points as far as possible. The man's last memory before the injury is usually a reliable point for the retrograde amnesia, but there are sometimes difficulties. Recollection is influenced by factors apart from the injury, such as the significance for the individual of events in the preceding period. Were they events worth remembering? Dull or exciting? Commonplace or unusual? The estimate of retrograde amnesia to be of real value should be accompanied by reference to these points. The estimate of post-traumatic amnesia needs similar details of circumstance. Moreover the observer must decide whether his end-point will be the first memory after the accident, or the beginning of continuous awareness. Often these correspond, but when they do not, when the first memory is, so to speak, an island, the beginning of continuous awareness of the surroundings is the safer guide. It would be convenient if all neurologists would agree to use this measure in stating the duration of post-traumatic amnesia.

In relation to the measurement of post-traumatic amnesia, it is important to know whether or not the patient has been given morphia, especially in cases in which the total duration is a matter of hours.

Type of headache.—We next want the story of the patient's symptoms up to the time of our examination, in relation not only to the sequence of time, but the sequence of events. We want to know, for example, not only whether he had headache and what kind of headache, but when he first had it, how often, and under what conditions it was worse or better. What was the effect upon it of having visitors, of sitting up, or first getting out of bed? All these questions apply equally to giddiness, and to the state of thought and feeling. If he was depressed or anxious, when and in relation to what circumstances, reflections or anticipations? What has his attitude of mind been towards the accident and its aftermath and what have been the stages in the development of his present attitude? This leads naturally to the analysis of his present complaints. Subjective symptoms are many and important.

The more experience I have of traumatic headache the more difficulty I have in dividing it into clinical types. There is, it is true, a localized variety, usually in the neighbourhood of the site of injury, intermittent, short-lived, sharp or throbbing, and related to physical effort or change of posture. This, when present, is highly characteristic of local injury, but it is uncommon in pure form. As another extreme example there is the continuous,

dull, generalized headache, unrelated to any circumstance. Either type may be encountered in individuals whose injuries have been of comparable severity, whose symptoms in other respects are the same, and who are apparently of the same constitution and disposition. Between these extremes there are all kinds of mixtures.

Knowledge of the circumstances which induce or relieve headache, and of the symptoms associated with it when it is present, is generally more helpful in differential diagnosis and treatment than the character of the headache. I would take as examples headache induced by continued though mild physical effort, such as walking, and associated with sensations of fatigue; headache similarly induced by continued mental effort and accompanied by a feeling of mental fatigue; headache induced by an unusual degree of stimulation of one of the special senses, such as noise or light; headache associated with mood disturbance, such as irritability or depression. Headache may be prominent after a brief amnesia. It may be absent after a prolonged amnesia.

If the amnesia has been prolonged it is useless to rely upon the patient's statement at a later date that he has never had any headache. It is not uncommon for a patient to have complained of severe headache during the amnesic period. It is therefore important that the observer of the earlier stages should record in the notes the presence, *or absence*, of headache during this phase. If a patient has reached the ambulant, or chronic, stage without headache it does not follow that he will continue to be immune. It is by no means uncommon to find a man beginning to complain of headache when, it may be several weeks after the injury, he is exposed to additional stress, and this is especially apt to occur when the exposure is sudden. It is important therefore when a patient has been headache-free up to the time of examination to know under what conditions of mental and physical stress this freedom has been preserved.

Dizziness.—A high proportion of patients in the stage under discussion complain of giddiness or dizziness. Of these a small proportion only describe true vertigo. Thus, out of 1,020 cases of closed head injury in which the symptom was inquired for, it was found present in 82. In 29 of these the vertigo was associated with deafness of middle or inner ear type, dating from the injury, and in 4 others there was a history of bleeding from the ear, or tinnitus in the early stage. This leaves 49 in which there was no evidence of aural damage. Two of these had damage to the 7th nerve, suggesting a fracture involving the petrous bone. In the remaining 47 cases there was no evidence pointing to the labyrinth as the probable site of injury. Of these 47 cases it is interesting to note that in 7 there was a history or presence of diplopia and in 2 others nystagmus was recorded in the early stages, symptoms indicating the probability of brain-stem injury.

There remain 38 cases, nearly half the total number with vertigo, in which no evidence either of aural or brain-stem lesion was forthcoming. We may, however, assume that vertigo in the true sense is evidence of damage to the vestibular sense organ or its central connexions. It is important evidence of organic damage and I suspect that if inquiry as to the presence or absence of deafness and tinnitus on the one hand, and diplopia and nystagmus on the other, were more rigorous in the early stages, we should have fewer cases in which corroborative evidence of labyrinthine or brain-stem injury is lacking.

Generally the complaint is not of true vertigo but of a transient disturbance of balance and often of the visual sense, experienced on stooping, or rather on rising from the stooping posture. This is probably due to a defect of vasomotor adjustment, and in the light of the recent experiments of Denny-Brown and Ritchie Russell (1941) may result from medullary concussion. It is a common constituent of the post-traumatic syndrome.

There are other varieties of dizziness which are less easily placed. I would draw attention in particular to one which is often described as a "black out". The onset is sudden, there is dimness of vision and a sense of insecurity of balance which may result in falling, without any description of true vertigo. Consciousness is often momentarily disturbed and may be lost. The main features of these attacks are syncopal rather than epileptic. Nevertheless, in some cases after repetition there is a transition into epilepsy.

An officer, aged 22, was injured in an accident on 7.10.39. Retrograde amnesia a minute or two, post-traumatic —forty-eight hours. He sustained a longitudinal fissured fracture of the left parietal bone and abrasions and contusions of the right chin, nose and left forehead. He found on recovering consciousness that he had anosmia and diplopia and suffered severely from headaches in the first three weeks. Within three months of his accident he was back on light duty with only occasional headache. The anosmia had persisted. The diplopia had recovered. Shortly after he returned to duty he began to have attacks which he described as "muzziness", "you can't think as clearly as you would like, your hands get sort of clammy". It came on gradually and faded gradually after five to ten minutes. No spinning or sense of movement in space, and no feeling that he would lose consciousness at all. He paid little attention to these attacks, which were infrequent, but on 27.5.40 he had an attack beginning in this way in which he lost consciousness for five minutes.

There was no history of epilepsy in the family. He himself, as a child, after running on a hot day, had once fainted for a few minutes.

The E.E.G. was normal.

He was retained in the Service in a restricted category and in January 1941 was admitted to another hospital on account of frequent attacks in the past three weeks of loss of consciousness. These would be preceded by a feeling of depression for one to three hours, together with drowsiness. He would then suddenly look very pale and become unconscious for several minutes.

The diagnosis made by experienced observers was that of epilepsy.

Mental disability.—Complaints of mental disability are, as a rule, classifiable under two headings. The patient tells us that in certain respects he is unable to perceive, remember and think as quickly and clearly as he could before the injury; and he says that somehow he feels different. Both kinds of disturbance are usually present in the same case.

There are many variations of this central theme, some more characteristic of brain injury than others. On the intellectual side, inability to enjoy reading for lack of power to hold the thread of the story, difficulty in grasping the war news, forgetfulness of small things, are significant. In the sphere of feeling the most typical complaint is of loss of interest and liveliness, but nervousness, depression and irritability are common. There are also cases in which the mood is elevated and activity increased. This patient often complains of nothing. His beaming smile and confidence of his own fitness are disarming and, at the same time, significant.

It is generally recognized that the family and personal history are important in assessing disability and guiding treatment. It is essential before we sum up the case that we should have at our disposal the main facts of the pre-traumatic personality and intellectual level, and know whether beyond this there are possibilities of an inherited and latent disposition to mental disturbance of a kind which may be precipitated by injury. There is as a rule more to be gained from this source than from a protracted neurological overhaul and the time available for examination should be distributed in accordance with these practical values.

Neurological Examination

By the time our post-contusional patient is examined he is unlikely to show any abnormal physical signs. Nevertheless, routine examination may occasionally reveal something unexpected and important. Of such signs anosmia is the commonest. If this is complete it will have appeared in the patient's complaints as inability to smell and taste, but there are many cases in which it is not complete. Bilateral anosmia may be present in a man who is yet able to distinguish flavours reasonably well. Unilateral anosmia as a rule passes unnoticed by the patient until it is specially looked for. Our usual methods of testing are, of course, crude, but when there is inability to distinguish test odours, without local obstruction or inflammation to explain it, and with a previous history of normal capacity for smell, we may presume that the disability is the result of the injury.

There are two facts in this connexion which are perhaps not generally known. One is that traumatic anosmia is not uncommonly associated with occipital fracture. The other is that it may result from an injury without evidence of severe generalized cerebral disturbance.

Captain D. F., aged 27. On 20.11.41 at a sing-song he was attempting to seat himself on a mantelpiece when he slipped, falling on his back. He does not remember hitting his head, but probably lost consciousness momentarily. He got up, but felt dazed and went to bed. On waking next morning he had a " thick head ", but no pain. He went on duty, which involved a long journey by car, during which he developed generalized throbbing headache, spreading down the back of his neck, nausea and eventually vomiting. He abandoned his journey and was admitted to hospital where meningitis was suspected. Lumbar puncture revealed a heavily blood-stained fluid with yellow supernatant fluid, and the diagnosis was altered to subarachnoid hæmorrhage. Headache and vomiting continued for three days, after which he rapidly improved. He found, however, that he had lost his sense of smell and could taste nothing in his food but sweet and bitter. He was transferred to a hospital for head injuries on 11.12.41 feeling well, save for occasional slight headache on reading. The positive findings were complete bilateral anosmia; a fine fissured fracture of the occipital bone in the mid-line running to the foramen magnum, and some low voltage 1 to 2 a second waves in both occipital regions in the E.E.G. There can, of course, be no doubt that the subarachnoid hæmorrhage was traumatic.

In a series of 1,020 cases of closed head injury, anosmia attributable to the injury was found in 76; bilateral in 62 and unilateral in 14. Of the bilateral cases 30 complained of their inability to smell and 16 of these also complained of inability to taste. Bilateral anosmia in this series was associated with X-ray evidence of fracture in 42 out of the 62 cases, the situation of the fracture being most frequently frontal (26 cases), and next most frequently occipital (12 cases), usually a fissured fracture of the occiput running into the foramen magnum.

A lesion of the optic nerve may have been missed if the field defect is small. Visual acuity therefore should always be examined. Lesions of the infra-orbital and supra-orbital nerves are also not uncommon in fractures involving the roof of the maxillary antrum, or frontal bone, and may be better evidence of fracture than X-ravs.

Dysphasia, hemiparesis or sensory defect, if they exist at this stage, will almost always have been detected and should have been recorded in the earlier and grosser stage. Signs of slight pyramidal damage may sometimes be found when least expected. Homonymous visual field defects will occasionally be missed if the method of testing by confrontation is omitted. Perimetry seldom yields anything of value if confrontation tests carefully executed are negative.

These observations will serve to indicate that the neurological examination at this stage should be intelligently guided. To include examination for dysphasia or dyspraxia,

perimetry or complete sensory investigation in the routine is a waste of time. On the other hand, failure to examine the sense of smell is a serious omission.

Psychiatric Examination

Intellectual impairment or personality disorder may be evident at the first examination and, if so, will of course be assessed against the estimate of the pre-traumatic state made from the history. The total situation must be taken into account, including the individual's adjustment, responsibilities, plans and ambitions before the accident and his reaction to the change in his environment and prospects following the injury. In many cases, however, a period of observation is essential before a just assessment can be made. This is equally true of civilian and Service patients. In the former the influence of over-anxious and over-sympathetic relatives, in the latter the inclination to exaggerate symptoms in order to evade unpleasant duties, may obscure the clinical picture at the first interview.

Special Investigations

X-rays.—By the time our hypothetical patient comes under neurological observation his skull will almost certainly have been X-rayed, but it is by no means so certain that the report he brings with him will be accurate. Common faults are inadequate pictures, misinterpretation and incomplete description. I suggest that radiologists should agree that when there is question of a fractured skull a standard series of pictures should be taken.

It is equally important that in X-ray reports the extent of the fracture should be described accurately, and with special care in the case of fractures running into, or close to, the accessory air sinuses, and that whenever there is any doubt as to interpretation this should be stated. The distinction between a small linear fracture and a vascular channel is notoriously difficult at times. In such cases the radiologist is perhaps unduly inclined to give the patient the benefit of the doubt, and it is not very uncommon to find a fracture reported when the final verdict decides that none exists.

Examination of the cerebrospinal fluid.—Lumbar puncture very seldom reveals any abnormality of pressure or constituents in the type of case under consideration. It may provoke severe headache in a patient who is on the mend and so impair confidence. It is therefore better omitted from the routine and reserved for the exceptional case in which there are clinical grounds for suspecting abnormalities of pressure.

Air encephalography.—What has been said about lumbar puncture applies with even more force to air injection, which should be reserved for cases in which there is gross evidence of organic cerebral damage from mental or physical examination. In such cases a lumbar or cisternal encephalogram may provide evidence which is of considerable value in pathological interpretation and assessment of prognosis.

Electro-encephalography.—The value of the E.E.G. in post-contusional states has been described so clearly and so recently by Denis Williams (1941) that I shall not recapitulate his findings. In about half the cases showing the chronic post-traumatic state he found an abnormal E.E.G. as compared with a figure of 8% by the same standards in a control group. The E.E.G. must now be regarded as an essential part of the special investigation in any case of severe or moderately severe head injury seen in the later stages. Its value will then be much enhanced if there is a record for comparison taken in the early stages.

Differential Diagnosis

Differential diagnosis in the stage which I am considering is seldom difficult if the record of the earlier stages is adequate.

As to the distinction between the physiogenic and the psychogenic factors in a given case, they appear in most cases so closely intertwined that to separate them is unnatural. I am thinking, of course, of the case in which there is no doubt that organic cerebral damage has occurred. That a man with a hurt brain should have a disturbed mind is to be expected. It is equally to be expected that this disturbance will affect his capacity for adjustment as a whole. What then follows must depend upon the psychological situations to which adjustment is called for. The disorder of function is related not merely to any set task of the moment, but a continuous series of adjustments. This is why our formal psychiatric tests are of relatively little value in assessing disability. We need to get inside the man as far as possible, looking back into his past and forward into his future. Even so, it is often impossible to measure disability except by putting a man to his old occupation for a continuous period of some weeks and seeing what transpires.

It will be understood from what I have said that I regard the practice of dividing the post-contusional cases into two groups, labelling the one organic and the other functional, or neurotic, as unprofitable and misleading.

For convenience of description I suggest that it would be better to use the ordinary psychiatric headings with slight modification, e.g. Brain Injury with Intellectual Impairment, Brain Injury with Depression (or other affective disorder), Brain Injury with Hysteria, Brain Injury with Psychopathy.

Treatment

There should be no delay once the examination is completed, in giving explanation and reassurance. In giving this we had best be truthful. We shall then be obliged to admit that headache, dizziness, difficulty in concentration and feelings of nervousness and depression are often slow to disappear; but we can say, at the same time, that symptoms of this kind seldom prevent a man for long from returning to his usual occupation, provided that he is patient and will make the best of things. We can dispel fears of insanity, and it is surprising how common these are. We can, and should, do a great deal more than this, of course, in the way of psychotherapy, which has a place in the treatment of every case of this kind.

The ideal atmosphere for treatment at this stage is that of a convalescent hospital at which there are well-organized departments for occupational therapy, physical exercises and indoor and outdoor games. The daily routine should be planned with suitable spells of compulsory rest in the early stages, and a reasonable allowance of free time. For Service patients the problem of disposal naturally looms large and this has close relation to treatment. Men who are to be invalided should be separated, as soon as the decision is made, from those undergoing rehabilitation for return to duty. When there is a probability that a man's category will have to be reduced, the sooner the decision is made the better, in order that he may know what lies ahead of him.

There is much ground for optimism in the treatment of post-contusional states, especially in young people. We see many cases of complete recovery after a post-traumatic amnesia of many days, or even weeks, but we should be wrong to take these as our standard. If we do so we shall be promising the majority more than they will get, and asking of them more than they can give. The results often are disillusionment and resentment. These two symptoms are often prominent in the post-contusional state and hard to get rid of once they are set. They are symptoms therefore which need to be nipped in the bud. Early and accurate prognosis are indispensable if this is to be done. Ideally we should wish to be able to tell the patient that in so many days, weeks, or months, he will be symptom-free, or fit to return to his occupation; or if there is no such good prospect ahead, to prepare him to restrict his activities and make the best of his disability. In fact, we are, I submit, all too doubtful in many cases of what the future holds, and for this reason the tone of our encouragement is often a little flat, or it may be sharp. Inquiry into the factors which influence the prognosis of brain injury is therefore most desirable. It must be detailed and extensive. Long term follow-up is essential.

We are at present, I think, too much inclined to assess prognosis in terms of those facts which are most easily ascertained. We have learned to discount fracture to a great extent, but there is a tendency to lay too much stress on the presence and duration of traumatic amnesia. It is well known, of course, that a man may suffer a severe localized cerebral injury from a penetrating wound without any loss of consciousness. This is rare in cases of closed head injury, but does occur. A man may, for example, suffer a permanent and totally disabling aphasia from blunt injury without having lost his senses. In a case without focal symptoms, however, the absence of amnesia is generally good ground for a satisfactory prognosis. It is much harder to generalize with regard to the duration of amnesia, when present. In a series of Service patients with closed injuries, the numbers of patients with different durations of post-traumatic amnesia who were invalided or returned to duty have been tabulated. It must be observed that the cases providing this material were a selected group. Most of them had been transferred to a Head Injury Centre because they were doing badly. Moreover, the conditions of duty to which they had to return were exacting as compared with the more flexible conditions of civil life. This, however, for purposes of observation is an advantage, since it may be assumed that the conditions for every patient in the series were comparable, and that the man who relapsed did so because he could not carry the load as well as the others, not because he had to carry a heavier load.

Post traumatic amnesia	I Number of cases	II Original disposal		III Proportion of original number invalided later	IV Total invalided
		Duty	Invalided		
Less than 1 hour ...	210	81%	19%	4%	23%
From 1-24 hours ...	302	78%	22%	7%	29%
From 1-7 days ...	216	71%	29%	9%	38%
More than 7 days...	143	63%	37%	11%	48%

It is apparent from column II in this table that in the assessment of prognosis in a certain age-group and in relation to fairly well-standardized occupational demands, the duration of the post-traumatic amnesia is of value. The longer the duration of the amnesia, the less likely is it that the patient will attain a degree of recovery which justifies the decision to return him to duty. Column III brings out another point. The figures record the percentage of the total number of cases in each group in which a follow-up showed relapse and subsequent invaliding. They show that of the men who had been judged fit for duty after rehabilitation (which included heavy physical training), a proportion were unable to stand up to the demands of Service life and that liability to relapse, or falsification of a good prognosis, progressively increased with the duration of the amnesia. Putting it another way, in men who have apparently recovered from the effects of their head injury, the longer the duration of the amnesia, the greater is the probability that residual defects of cerebral function will be revealed by the crucial test of return to what is, for the Service patient, a normal mode of living.

Column IV shows the total percentages invalided in each group, including the relapses. This reveals that if the duration of amnesia, without consideration of other factors, were to be taken as the sole criterion of prognosis, the expectation of successful return to duty for those with an amnesia of less than an hour is 77%, as compared with 52% for those with an amnesia longer than seven days.

From whatever angle these figures are viewed, therefore, the value of the duration of the post-traumatic amnesia as an index of prognosis is apparent. It is, however, equally apparent that the duration of amnesia is not the only factor which counts in prognosis. For example, if we were called upon to give an opinion upon the prospects of return to duty for a man who had recently emerged from a post-traumatic amnesia of ten days' duration, without examining the patient, and without reference to any other details of the case, on the basis of these figures, whichever way we decided the chances of our being right or wrong would be about equal. Taken by itself, therefore, the duration of the amnesia does not carry us very far on the road of prognosis. One patient with an amnesia of two or three weeks may be back at duty within four months of his injury, and succeed; another with an amnesia of less than one hour may not get back to duty at all, or having done so, may fail. If, therefore, we are going to make use of the post-traumatic amnesia as a yardstick by which to measure the severity of the injury in terms of prognosis, we should use it with a good deal of caution, and with a keen eye for all the other factors which may weigh the balance in one direction or the other.

Success in the treatment of closed head injuries—and I am thinking now especially of success as measured in terms of the shortest possible period of invalidism—has been hindered in the past by the traditional acceptance of fixed rules, such as that which imposed three weeks flat in bed for every patient with loss of consciousness, however brief, or that which necessitated so many weeks' absence from work after a fractured skull. It would be a great pity if, at this stage of our knowledge, we should enslave ourselves to fixed rules based upon the duration of post-traumatic amnesia. I have stated elsewhere (Symonds, 1941) reasons for supposing that the duration of the post-traumatic amnesia is mainly dependent upon a generalized disturbance of cerebral function, which is reversible. A long duration of amnesia, therefore, is compatible with complete—and rapid—recovery after clear consciousness is recovered. Inasmuch, however, as the duration of the amnesia is a measure of the severity of the generalized disturbance, it is also a measure of the severity of the blow. The greater the severity of the blow, the more likely it is to have caused local structural damage with long lasting or permanent effects, in addition to the generalized, reversible disturbance of function. It is to be expected, therefore, that symptoms of coarse cerebral damage will be observed more often after a long amnesia than after a short amnesia. During the period of clouded consciousness the most important of these symptoms, those indicating mental impairment, are masked. Therefore, it is not until some time after the patient has recovered clear consciousness that the extent of the more lasting effects of the injury can be gauged. It follows that examination of the patient, and especially the examination of mental function, *after the period of amnesia is over*, is a truer guide to prognosis than the duration of the amnesia itself. These views have been confirmed by the impressions gained from the experience of the past two years, though they have yet to be subjected to the analysis of factual data collected from a large series of cases.

Meanwhile I deprecate the use of such a table as that proposed at a recent discussion before this Section (Cairns, 1942) in which the duration of the post-traumatic amnesia is set out in relation to the shortest time in which ability to carry out full work may be expected to return. For example, the first group taken is that in which the post-traumatic amnesia is from five minutes to one hour, the minimum period of disability for full work being stated as four to six weeks. Certain qualifications are made, but there is no mention of symptoms suggesting focal structural damage, which I believe are of greater

importance than the duration of the amnesia. If the working rule provided by Cairns were followed there would be liability to error in two groups of cases. The first, which is numerically more important, comprises those in which, after a momentary loss of consciousness, there is mild confusion and automatism with amnesia, often for more than five minutes, and not infrequently for more than one hour, and with complete recovery in the course of a few days. I have seen many such cases in civil practice, and have no doubt that many such occur in the Services which are never seen at Head Injury Centres. The second, and smaller group, is represented by the man whose amnesia is of less than five minutes' duration (it may be nil), who suffers prolonged disability, possibly for several months, on account of localized headache, intellectual impairment, or personality change, probably as the effect of localized cerebral contusion. This group of cases Trotter rescued from the dumping ground of traumatic neurasthenia. It would be a retrograde step to put them back there, yet there is danger lest too close an adherence to the rule of the duration of the post-traumatic amnesia may lead to the assumption that disability beyond the limits of this rule must be psychogenic.

I am aware that I have discussed prognosis at some length, whereas it is not included in our title. I make no apology for this, for I believe that many of the symptoms which we have to treat arise from the uncertainty in the patient's mind about whether he will ever get rid of his headaches, or when he will get back to a normal existence. I hope that the material accumulated by the head injury centres in this country will enable us to get rid of some of the uncertainty in our own minds about the answers to these, and other questions.

REFERENCES

CAIRNS, H. (1942), *Proc. Roy. Soc. Med.*, **35**, 299 (Sect. Neurol., 5).
DENNY-BROWN, D., and RITCHIE RUSSELL, W. (1941), *Brain*, **64**, 93.
SYMONDS, C. P. (1940), in "Injuries of the Skull, Brain and Spinal Cord", edited by Brock. London.
WILLIAMS, D. (1941), *J. Neurol. & Psychiat.*, **4**, 131.

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Dr. Aubrey Lewis: During the first few weeks after a head injury it will often be necessary to decide on the causes and pathology of the mental condition. That it is mainly exogenous will be concluded when there has been clear evidence of cerebral damage (I suppose that the term "contusion" is not going to be applied unless there is such evidence) and where some "organic" symptoms have followed close upon the injury. Direct visual evidence of cerebral damage, such as the surgeon may have, will of course often not be forthcoming, and the evidence will then be only of disturbed cerebral functions, among which consciousness will be the most important. The signs of a damaged brain, apart from the focal ones revealed by neurological examination, are much the same whatever the lesion: various degrees and combinations of impairment of memory, grasp, orientation, perception, thinking, affect and spontaneity. When an exogenous brain syndrome, following trauma, has been recognized, the question of ætiology has not been disposed of. The patient's delirium, for example, may be a straightforward delirium tremens far more dependent on his long-standing alcoholism than on his recent head injury; you can call that differential diagnosis, if you like, but it is better to consider it as ætiology since both the alcoholism and the head injury have probably contributed to it. Differential diagnosis is too prone to insist on absolute verdicts between more or less incompatible claims. There are several physical causes to be reckoned with, in acute post-traumatic mental states: alcohol, infections, presenile and senile conditions, cerebral vascular disease, G.P.I., tumours and epilepsy. Besides these diseases, which may occasionally play a large part in causing the acute mental disturbance following head injury, there are constitutional causes predisposing the patient to this or that type of disturbance. His fatuity and euphoria, for example, may be more eloquent of his hypomanic disposition than of a destructive lesion of his brain; his apathy and lack of initiative may be akin to the depression he formerly experienced after a bereavement or an attack of scarlet fever, and may have little to do with his frontal lobes; some patients exhibit schizophrenic syndromes as soon as they have recovered consciousness, others pass by stages through stupor and confusion into schizophrenia. It would be inappropriate to consider here how the catatonic stupor or excitement released by cerebral trauma may be distinguished from the exogenous traumatic syndrome coloured by schizophrenic trends. In either case, however, the previous history of the patient, and especially his personality may be an important guide.

So much for the early conditions, appearing during the first few weeks after injury. There is, of course, no clear distinction between early and late post-traumatic syndromes.

But hitherto I have been considering those developing while the patient is still indisputably suffering from the effects of cerebral damage. If the evidence of damage has been slight, and the period of unconsciousness in particular quite brief, this stage is soon over, though it may be assumed that by a contusion something more than a mild or trifling concussion is meant.

The conditions seen during the later stage can be divided into three classes: (1) the semi-chronic or chronic organic syndrome (usually a Korsakow amnesic one or a dementia); (2) the semi-chronic or chronic insanity, usually a schizophrenia; and (3) that common, dubious, psychopathic condition—the bugbear of the clear-minded doctor and lawyer—post-traumatic neurosis and personality disorder. It includes the "minor contusion syndrome" of Symonds, the "psychasthenia" of Mapother, the "traumatic psychopathic constitution" of Ziehen, and the "post-traumatic personality disorder" of American writers. Uncertainty about it turns mainly on the question: Is it due to structural damage or is it psychogenic? The insistence upon this is understandable, but fallacious; understandable, because the somatic pathology of any disorder is of prime importance, and because so many social issues such as attributability and pension rights depend upon the answer to the question; fallacious because it ignores the real state of affairs at present, and asks us to say "Yes" or "No" to a question often unanswerable in that form.

The question presupposes that exclusively physiogenic and exclusively psychogenic cases can occur, and that every case will be at least predominantly psychogenic or predominantly physiogenic. To substantiate such a view, the criteria of psychogenesis or of physiogenesis must be clear and demonstrable. But they are not. Physical damage to the neuraxis can produce all sorts of mental symptoms, including neuroses and personality disorders: encephalitis lethargica is a convincing example of this obvious truth. The ordinary features of an exogenous mental syndrome may be totally lacking. It is therefore impossible to infer whether a mental syndrome is physiogenic or not by study of the symptoms presented in it. There is nothing characteristic of the syndromes thus produced by certain kinds of structural damage, to enable us even to group them together as of structural origin. Are we then to conclude that a condition is physiogenic whenever we can prove existent cerebral damage? Scarcely, because the brain may have adapted itself, as we know it can, to this lesion, and persisting disturbance of function may not then be attributable any longer directly to the tissue damage. Moreover in a large proportion of the cases in question there is no evidence of existent physical damage to the brain.

It could not be said that we are on safer ground in settling the criteria of psychogenesis. It is notoriously easy to find psychological causes if you look hard enough. You can find them in patients with tumours and disseminated sclerosis and carbon monoxide poisoning and all sorts of organic diseases. The adequacy of the psychological motives to account for the symptoms can be so much a matter of personal opinion—one doctor disagreeing entirely with another doctor—that it is hardly to be thought of as a useful criterion in any dubious case. For rather similar reasons the response to a change in the situation or to some psychological device or treatment can be deceptive and ambiguous: even a dramatic change can be produced, say, by hypnosis in the symptoms of a patient with disseminated sclerosis.

These arguments might perhaps be dismissed as casuistry since there are cases where no one is in doubt as to the mainly physiogenic or mainly psychogenic nature of a neurotic illness. But it is because of the lack of any unequivocal and agreed criteria of physiogenesis and psychogenesis that we are so often in a dilemma in diagnosing a patient with a post-contusional neurotic syndrome.

I believe that we have no unequivocal criteria, no final distinction, between physiogenic and psychogenic because the search implies a dualism which is not there. Focal brain damage may produce characteristic disturbances of function, usually seen as neurological signs: gross widespread brain damage may produce disturbances of function, recognizable and characteristic of exogenous mental syndromes (though even these may be closely mimicked by affective disorders and by schizophrenia); but less acute and coarse disturbance may produce nothing that could not also be produced in a man with an intact brain, exposed to stresses of another sort. The patient, as a wholly integrated human being, deals with what happens to him in ways that are determined by his hereditary endowment and previous experiences: if he sustains an injury to his head, his behaviour at any subsequent stage cannot be thought of as simply the sum of his normal functions plus the reduced or altered functions due to this destructive lesion. His behaviour at every stage is a reaction to an existing situation in which his symptoms at the time, his financial, social, domestic and other difficulties are elements; the form of this reaction will obviously be determined by what has happened to him up to now. It is therefore in principle a plastic response, not a fixed one. The physician who concentrates on

the cerebral damage is treating behaviour as though it were a neurological sign, constant and always referable to some local place of origin. If at the other extreme he concentrates on the environmental factors and the psychological reaction, he may be ignoring the most important aspect of that recent happening which has left its mark on the patient's brain, creating perhaps fairly rigid symptoms (like diplopia, dysphasia or even a headache) and making a pattern of behaviour from which he can now hardly depart.

If such a point of view is held, there is no sense in supposing that one must always decide whether late post-contusional syndrome is physiogenic or psychogenic. There will always be some of the latter in the causation, there may be quite a lot of the former. If one can appraise the physiogenic residue by neurological examination, or electroencephalography or other special device, of course one will do so. Even if it is extensive one will not treat its disturbing effects on function as irremediable; any more than one wants, after appraising the extensive psychogenic side, to psychoanalyse it or regard it as original sin. Before speaking of treatment, however, there is more to be said about diagnosis. Some of the symptoms may be obviously hysterical; sometimes the whole syndrome cries out hysteria. Even so it cannot safely be concluded that there was no structural damage to set these works going: there may have been. The headache, giddiness, lassitude, forgetfulness, insomnia, may differ in time of occurrence, degree and other characters from these symptoms as met with in the earlier stages of post-contusional disorder when the symptoms may be assumed to be chiefly due to structural damage. It would be misleading to infer that they are now, therefore, wholly a motivated construct; they may be hypochondriacal exaggeration of existing physiogenic symptoms. No one would deny that a person free from detectable neurotic predisposition may, after a head injury, become irritable, easily tired mentally and physically, depressive and apathetic, because of cerebral damage. But for one such easy case there are half a dozen or more, difficult ones, by no means clear-cut, and even in this easy case a little carelessness in the handling may result in prolonging or fixing the disability. Such carelessness arises from preoccupation with the antimony—physiogenic or psychogenic—which I have been deprecating for clinical purposes. Research into the problem is a rather different matter.

Assessment of physiogenic damage.—Goldstein and other workers have tried for a long time to use psychological tests to detect physiogenic damage to the brain. Pfeiffer's description of a long array of psychological tests occupied fifty pages of his monograph on mental disturbances after brain injuries in war, and in the twenty years since that was written, a great deal more has been done. It would therefore be impossible to review the matter in any detail. The bulk of recent work has been much influenced by Goldstein who emphasizes the disturbance in "categorical", i.e. conceptual thinking. Tests for conceptual thinking do not, however, cover the available methods. Babcock devised a method of detecting and measuring deterioration which relies on a discrepancy between present capacity and presumptive previous capacity estimated by a vocabulary test. Changes in personality due to head injury are referred by Goldstein to the conceptual difficulties, but may be studied in their own right, as by the Rorschach procedure. Disturbances of memory have been investigated, as in Zangwill's study of the Korsakow state.

The work done in this field by E. L. Trist and his co-workers at Mill Hill has taken account of most of these methods, and attempted to combine a number of different tests in a set that could be administered in a short time, say, half an hour, and would be clinically useful. No single test suffices to pick up deterioration because it is not a matter of independent functions such as memory, attention or intelligence which may each be separately affected. The tests used were modifications of the Shipley vocabulary, Dworetzki pictures, Wechsler's similarities, a sorting test using shapes, Weigl's colour-form sorting test, and Kohs blocks: other tests such as Vigotsky blocks, Bolles' and Halstead's sorting methods were also examined. So long as the investigation was limited to persons with known cerebral damage, all was well: tests revealed the expected disturbance. This was true of the group of treated general paralytics who were selected as the most satisfactory analogue to the late post-contusional patient: the cerebral damage in them being certain but non-progressive, and often no longer clinically detectable. But when the same set of tests was administered to a group of neurotic subjects in whom there was no reason to suspect any structural damage, some of them behaved very much as did some of the patients with G.P.I.: the same was true of "normal" subjects tested, though this was less evident if one took the set of tests as a whole than if one looked at individual tests. To put a complicated matter briefly, it has become evident that when the diagnosis of organic damage is clinically doubtful, psychological tests cannot as yet be relied on to supply an unequivocal answer: there is no psychological Wassermann reaction here, no skin test, X-ray or blood picture to settle the vexed question. Because

the psychological tests do not settle the question of cerebral damage they are not to be thought useless. There are many cases in which the results of these tests are such as could only be yielded by cerebral disease; and in any case, besides the diagnostic issue it is often necessary to know exactly what functions are impaired and to what degree, so that suitable work and treatment may be offered the patient, and the rate of his improvement measured: for these purposes the psychological tests mentioned and others of a more special aptitude-measuring kind are indispensable. These tests, in short, are in the same relation to our routine clinical investigation of memory, grasp, &c., as the standardized intelligence test is to our impression of a man's intelligence: precautions are necessary in interpreting the standardized measure just as in interpreting our rough clinical findings. The validated set of tests, with its items systematically considered and checked, is a more precise instrument, and its findings on different occasions can safely be compared so that one can tell if the patient is improving in particular respects.

There are many other psychological tests, less concerned with the fundamental question of deterioration, that have been used in cases of brain injury. The most interesting and popular of these is the Rorschach inkblot—which is chiefly a personality test. The elaborate methods of interpretation and the obscure terminology of this test make it a rather esoteric affair, and I cannot pretend to be more than a proselyte at the outer gate. Piotrowski has laid down canons of diagnosis for organic cerebral disease by this method, and Harrower-Ericksen in Montreal has made easily understandable contributions to the same matter. It has been plain, however, in the work done by Miss Harvey, Trist and others at Mill Hill, as well as in Ross's paper, that what had been regarded as organic types of response to the test can occur in insane persons, hysterics and other psychopaths, and normal people of poor intelligence, without any organic cerebral affection. The position is, again, that these types of response occur commonly in organic disease but they cannot in a doubtful case, where there are psychiatric symptoms, be used as diagnostically decisive. For some aspects of the patient's personality, the Rorschach findings are illuminating, whatever the cerebral condition.

Survey of a Series of Cases

I have lately made a survey of a series of post-contusional states admitted to a neurosis centre. There were 64 patients in the series, all men, nine of them civilians and the rest soldiers. The form of the clinical syndrome displayed was diagnosed in the usual psychiatric terms, and a group of 64 patients taken for comparison from the other neurotic patients in the hospital. The selection of these was at random except that they were of the same sex, included the same number of civilians, and exhibited the clinical syndromes in the same proportion, as did the head injury cases. Thus there were 16 patients with conversion hysteria in each group, 2 patients with hysterical amnesia, 6 with a severe acute anxiety state, 14 with a chronic anxiety state, and so on. The number of cases, 64, may seem small, but the number of attributes in respect of which they were compared was nearly 150, and covered practically all the main points of psychiatric interest in each case. The main items are shown in Table I.

The points at which the two groups differed significantly (i.e. statistically so) were remarkably few. More men in the control group had been discharged Category E, had as adults shown signs of predisposition to mental disturbance, had been unsociable, weak and dependent, lacking in initiative, over-anxious, hypochondriacal or obsessional. More of them complained of pain (apart from headache) and anxiety symptoms; whereas the head injury cases included, as would be expected, more people who had been of stable, well-organized personality before their illness, and severe headache, fainting and irritability were commoner among them. But the differences in these respects were only on the margin of statistical significance, and it was evident that the head injury series was made up of very much the same sort of people (in family and personal history, intelligence, symptoms, response to treatment, and outcome) as the non-organic group.

It is clear that these post-contusional cases had been sent to a neurosis centre because some doctor thought they were of a particular type: they are not necessarily representative of the minor contusional syndrome, they will include a perhaps unduly high proportion of those whom the doctors referring them judge to have recovered from all physical effects of their trauma. They were, however, very good examples of the syndrome, clinically, and many of them had had very severe head injuries; where they differed from the average case, I think, was in the length of time that had elapsed since the injury, so that features of chronicity and habituation were prominent. However, the striking thing is that the long-standing, relatively intractable post-contusional syndrome is apt to occur in much the same person as develops a psychiatric syndrome in other circumstances without any brain injury at all.

TABLE I

	Head injury	Control.
Age		
15 to 20	2	5
21 to 25	16	15
26 to 30	20	24
31 to 40	20	12
41 and over	6	8
Service Patients Only		
Service Occupation—Skilled	12	15
N.C.O.	6	3
Category on Enlistment—Not A	7	8
Category on Discharge—E	15	25
Annexure A	15	9
Annexure B	11	10
Civilian Occupation		
Professional or Administrative	6	11
Skilled	18	10
Earnings		
£3 and Under	25	30
£7 and Over	5	2
Unemployment		
Much	7	10
Little	10	17
Work History: De-graded or unduly frequent changes	9	5
Duration of Stay—More than two months	9	16
Family History		
Psychosis	7	14
Neurosis, &c.	22	24
Personal History		
Upbringing other than by Parents	1	5
Upbringing Unsatisfactory	9	15
Education:		
Elementary—Poor	12	18
Secondary or Central	3	8
Higher	2	1
Sex Anomalies	6	12
Social Activity—Much	11	5
„ „ —Little	33	43
Past Physical Health		
Medium	18	27
Bad	2	4
Epilepsy	1	0
Previous Organic Disease of Nervous System	1	3
Previous Organic Disease Elsewhere	9	8
Previous Mental Health		
Symptoms in Childhood	17	17
Predisposed in Adult Life	18	28
Definite Illness	10	6
Previously Treated in Mental Hospital	1	0
Out-Patient Department	0	2
Private Doctor	12	12
Previous Personality		
Stable, &c.	36	25
Weak, Dependent, &c.	23	39
Delinquent	2	4
Inert, without Initiative	11	23
Rebellious	18	17
Suspicious	25	29
Cyclothymic	17	20
Schizoid	17	20
Hysterical	15	23
Anxious	30	44
Hypochondriacal	21	30
Obsessional	8	20
History of Present Illness		
Onset during Training	11	15
Exposure to Enemy Attack:		
Severe	20	8
Medium	11	14
Symptoms		
Somatic Anxiety	25	28
Headache—Mild	23	24
„ —Severe	25	6
Dizziness	27	21
Fatigue	24	28
Effort Intolerance	21	18
Dyspepsia	7	14
Fainting, &c.	13	3
Pain	7	29
Severe Tremor	3	6
Stammer	5	8
Enuresis	2	2
Sexual Anomalies	2	5
Anxiety—Mild	21	15
„ —Moderate	16	26
„ —Severe	8	8
Depression—Mild	28	24
„ —Moderate	15	16
„ —Severe	4	1
Apathy	20	11
Perplexity	4	10
Hypochondriasis—Mild	16	12
„ —Moderate	14	17
„ —Severe	0	4
Depersonalization	1	1
Hysterical:		
Motor	4	3
Sensory	16	12
Special Senses	6	1
Visceral	3	4
Dysmnesic	4	4
Obsessive, Compulsive	5	8
Dementia	7	2
Stress of Bombardment	15	12
„ Domestic, &c.	19	14
„ Separation, &c.	24	32
Treatment		
Discussion, &c.	39	34
Hypnosis, &c.	7	5
Narcosis or Insulin	1	2

Incidence of Head Injury in the Psychopathic Personality

This raises a further question—are people of psychopathic predisposition more likely to sustain a head injury than others? If gunshot wounds were the common form of head injury, the answer would certainly be "No". But while accidents on the roads remain so frequently responsible, a more careful answer is needed. I suppose everyone reading through a series of head injury records is struck by the way some of these patients seem to have been dogged by a malicious fate so that they have had two, three or even four head injuries in the course of five or ten years. It is less likely that one head injury predisposes you to have another than that some people are particularly prone to accidents because of some slowness of reaction in an emergency, defect of judgment, or other trait. Farmer and Chambers, in their industrial Health Research Board Report, No. 84, have shown convincingly that accident proneness is an important factor in motor drivers, leading to repeated accidents, and that experience in driving does not avail to alter the differences between those specially prone and others: moreover they found that certain psychological tests were done badly by those with a high average accident rate. It is not unlikely, therefore, that among those who sustain head injuries in road accidents there will be a higher than average proportion of predisposed, and perhaps neurotically unstable persons. This is not to deny that many who sustain head injuries have previously been well-adjusted, healthy people. It emphasizes, however, the need for looking into the previous personality of the man with a post-contusional syndrome and indeed paying at least as much attention to this as to the extent and persistence of cerebral damage.

The situation arising out of the accident has also to be considered, if the true ends of diagnosis are to be served, for this may be the most potent of all the causes of the post-contusional syndrome. I said earlier that the patient has to react at any stage to the existing situation. I would like to stress that this situation cannot be reduced to a few salient features, any more than personality and behaviour can. To single out the compensation side of it, or the chance it offers of escaping with honour from disagreeable duty, is to overlook a great deal. Often the desire to obtain money is construed by the doctor as the main motive in the patient's continued illness when cerebral damage no longer suffices to account for his symptoms. By no means all the non-physiogenic post-contusional syndromes are hysterical—depression and anxiety are conspicuous: nor are hysterics who claim compensation actuated only by this in the production of symptoms. Loss of employment, insecurity, and many other forms of social pressure are at work, not to speak of the hypochondriacal, anxious and other latent trends now set in motion by the severe threat to his life, his reason or his health which the patient believes he has sustained. His symptoms themselves form part of the situation he must cope with. We can see this easily enough in a man with dysphasia or squint—he obviously has to adapt to the disability—but we tend to overlook it when his symptoms are more of the psychological kind.

So much for the diagnosis of these common and often difficult cases—not so much diagnosis, I suggest, as appraisal of multiple causes, the doctor taking care neither to hunt the snark of physiogenesis to death, nor perfervidly to track the red herring of moral obliquity (" gold digging ", " scrimshanking ") to its lair.

Treatment

Treatment is more preventive than actual. The damage done by ill-advised treatment in some of these men could not be put right by a demigod. I would mention only what seem the essentials of preventive treatment: (1) To decide early what plan to adopt and, as far as possible, to adhere to it. (2) To let the patient know, as soon as may be, that he will, or will not, have such and such residual disability which will clear up, and that he need fear only so much incapacity, or none at all eventually. (3) To see that misguided relatives or friends do not tell him a highly coloured story of the accident, but that it is explained to him soberly and with due allowance for his amnesia and other symptoms. (4) Not to prolong the period of rest and inactivity, but to institute early some mild work or interests, no more exacting than his state warrants, and gradually to increase both the opportunity for activities and the incentive, taking care on the other hand to avoid overtaxing him to the point where frustration and " catastrophe reaction " could lead to an exaggerated concern over his disability. (5) To help him in any financial, legal or domestic embarrassments to which the accident has conduced: a skilled social worker is here most valuable. (6) To do everything possible to bring the phase of special examinations to an end, except in so far as they are necessary for assessing progress or deciding on special methods of treatment or disposal. I do not here refer to regular definite investigations (whether physiological like the E.E.G. or psychological, like the set of tests mentioned earlier) but to the repetition of X-rays, lumbar punctures, and other procedures which give the patient the impression that the doctors are not sure about him, that they cannot decide whether his brain, or his mind, is seriously damaged; worst of all is it when he passes from hospital to hospital, each repeating the investigations and perhaps reversing the diagnosis or the treatment advised at the previous one. These and other precepts are obvious enough, but they have often been flouted, heedlessly and harmfully, by the time the patient reaches the neurosis centre, at any rate, and I suppose head injury centres could tell the same tale. Much of this over-investigation must arise as I have said from mistaken concern over the question whether the illness is physiogenic or psychogenic—a question that is often the parent of muddle, though intended to bring light and clarity.

Rehabilitation

This subject was discussed three months ago before the Section.[1] A few points, however, call for brief reference. It was said in that discussion that rehabilitation will include occupation (diversional, constructive, and useful to the hospital), physiotherapy, and intellectual and recreational pursuits. There is no mention in this list of any special care for the patient's individual psychological problems. It is obvious that work and physical activities, games and reading all exert some psychological effect, that in this indeed their chief efficacy may lie; but the patient is an individual and unless his private difficulties and attitudes are given sufficient attention, the rest of the valuable routine may not avail to make him well. At different periods after his injury the importance of these psychological problems varies; the later it is, the more they control the illness and

1 *Proceedings*, **35**, 295 (Sect. Neurol., 1).

should determine its treatment. More explicitly, one might say that after severe or moderate cerebral contusion, for a while the patient reacts as an average human being whose brain has been damaged at certain points, rather than as a particular human being who is in a particular fix. Later on the individual difficulties and reactions overshadow the general, more or less common, pattern of disturbed cerebral function. But what I said earlier indicates that this needs qualification, and that the influence of the patient's constitution, his past, and his present circumstances must be reckoned with from the outset. It is not possible to reckon with them to good purpose unless one has knowledge of them. This knowledge, which may be obtained sufficiently from a relative of the patient, is in many cases all that is necessary and practicable in the early stages after his injury, and may be all that is necessary throughout; but there are some in the early stages for whom it is not enough, and it is hardly ever enough in those later stages when the "minor contusional syndrome" has asserted itself ominously.

In other words, you are unlikely to succeed in getting rid of the patient's symptoms if you can only surmise what factors are producing these symptoms; and even if your surmise is correct you cannot always deal with these factors by environmental adjustments (through the social worker and the relatives) or by indirect methods only such as those listed under work, recreation and physiotherapy. Direct psychological treatment is called for—not, of course, invariably. I think there is a good deal of misunderstanding about this. Psychological treatment of any sort is good or bad according to its appropriateness in the particular case and the skill with which it is conducted; harmful probing is, of course, unskilful, as superficial dabbing can be, or crude ploughing and plugging. Psychological treatment will not consist in a choice between the extremes—psychoanalysis or a casual chat. As Colonel Cairns put it in that discussion "no attempts at rehabilitation are likely to be successful unless the patient's anxieties and fears are assuaged and unless he is helped through the phases of depression and the other disturbances of feeling that so often beset him during recovery from head injury". These affective disturbances may turn on responsibility for the accident in which others were injured. I lately saw an Army dispatch rider with an obstinate post-contusional syndrome including pronounced hysterical features in whom it required much finesse and persistence in delicate inquiry, before one learnt that he was in great financial difficulties, that these arose out of his attempt to contribute from his scanty Army pay to the support of the orphan of a man on another motor bicycle killed in the accident in which the patient as driver had had his head injured, and that this in turn was linked up with the censure pronounced on him for the accident by the Army authorities, which he considered unjust—they had degraded him—so that he had a strong hatred of his Army superiors and what they stood for. All this tangle had to be cleared up before he could improve. Some of it was cleared up by simple and obvious measures, some only by addressing oneself to the sources of his guilt, depression and resentment. I am not suggesting that in most post-traumatic syndromes one needs to behave as though the patient was one's oyster, but that it is equally senseless to assume he is a clam. At all events, oyster or clam, he will be better for tactful discussion of "what is on his mind"; this will vary so much from patient to patient that general inferences, e.g. about the effects of industrial injuries and the Workmen's Compensation Act can have only partial validity for any individual. To avoid misunderstanding, I should add that I believe social factors to be more important than individual propensities in keeping these residual neuroses going—Dr. Russell Brain's figures illustrated this—and that social adjustment, like social measures of prevention, is the essential preliminary to any treatment and itself a more effective means of treatment than psychotherapy alone can be; but psychotherapy, however brief and simple, or however recondite, should never be conducted as a thing apart from social adjustment, occupation, and the other features now recognized to be indispensable for restoring, as for maintaining mental health. The trained psychiatric social worker is often the person who does most, by direct action, to bring about the patient's recovery from a post-contusional syndrome; but, for this, she needs the guidance of the doctor in touch with the patient's emotional and private problems, and the doctor will not be able to give it who relies solely on a well-ordered, progressively adjusted hospital routine of physiotherapy, occupation and other pursuits to do everything for the patient.

Occupation.—Here too the ground was so admirably covered in the previous discussion that there is no need for restatement. I would only stress that it is profitless to let a man do trivial or absurdly easy work at a stage of his illness when neither intellectual nor affective disturbance prevents him doing something more like the jobs of ordinary life. Occupational therapists sometimes allow the principles suitable for treatment of inert melancholics and semi-stuporose or preoccupied schizophrenics to operate in a different type of illness, so that it is thought a triumph if the post-traumatic patient labours dully and steadily at some dreary repetitive job, or now and again tackles in spurts

a task well below his actual powers. The patient is thought to be doing excellently when he applies himself assiduously to painting butterflies on glass or disfiguring wood with poker work mottoes. Many men with late post-contusional symptoms are content to loiter along in these pointless activities, which are as demoralizing as idleness. It is necessary at all stages to have the man doing something worth while; not of course flying too high and becoming upset or dispirited at failure, but not, on the other hand, accepting resignedly a low level of work, and aiming only at what would be exclusively leisure pursuits for him, e.g. rugmaking or raffia work. What I am advocating was tersely put by Dr. Brain when he said "occupational therapy should merge into therapeutic occupation"—but in some hospitals the rule that any occupation is better than no occupation seems still to be the high-water mark of aspiration.

There is no need to discuss at length the question of physiotherapy or of treatment of special disabilities such as dysphasia and epilepsy. As for the "intellectual and recreational pursuits", there is much to be said for making these unobtrusively part of the therapeutic plan. It would, of course, be foolish to tell the patient what he is to read, and to try to control every detail of his day; but if he is to have, let us say, as a soldier, some educational lectures while in hospital, let these have a bearing on real problems, stimulating his interest, but also providing incentives to counteract those which are perpetuating his symptoms. To illustrate this from actual experience would take too much time. There is, however, one still experimental instance of this which shows how education as commonly conceived and education as part of the psychological treatment can be combined: the doctor collects his patients in a group, talks to them a little about some familiar difficulty or misconception that often crops up when he is examining them alone or inquiring about their notion of their illness, and then invites them to ask questions. By this means prejudices and wrong attitudes can be to some extent corrected and the doctor's time economized. One of my colleagues, Dr. Jones, has used this method as an adjunct to treatment of a rather similar group (patients with cardio-respiratory neuroses) and has found it useful and economical. None of these methods is sufficient by itself.

The "demoralization", or psychopathic change in personality, that may follow brain injury, especially in children, is a more difficult business, though at bottom it is the same problem. I cannot say that I have seen outstanding success in the treatment of genuine instances of this; spurious instances are of course common and may do well. It is like the post-encephalitic behaviour disorders: you can palliate by training, but that is all.

A few words are necessary about the later forms of post-traumatic insanity. They have an incidence of nineteen per hundred thousand of the male population in the corresponding ages. The rate rises from 10 in the 20-29 age-group, to 16 in the 30-39 group, 20 in the 40-49 group, and 25 in the 50-59, 60-69 groups. This increased incidence as age advances cannot be accounted for by an increase in accidents sustained, but must be construed as another instance of how the ageing process is itself, with its reduced functions and loss of resilience, a very prominent cause of these traumatic psychoses. In short it is more an involutional or presenile disorder here than a traumatic one, and the age distribution very similar to that of presenile and other degenerative organic psychoses. Mayer-Gross and Feuchtwanger have dealt very fully with a series of post-traumatic schizophrenias, showing how diverse the factors and course can be. The persistent amnesia or Korsakow states and dementias are seldom uncomplicated by alcoholism, atheroma, senile or other somatic disease. It will depend on these other factors—constitutional or morbid—and not upon the injury itself whether the psychosis follows hard upon the accident or there is an intervening period of apparent recovery. This explains I think Mapother's observation that where schizophrenia or a paranoid syndrome supervenes after an interval of normality, the prognosis is worse.

The late post-contusional states are an exemplar of what social and preventive medicine means in the neuro-psychiatric field. The best the doctor can do may fall short because adverse genetic and social forces are at work which he cannot remove, and the effects of which he can only lessen. The least of his obstacles will often be the residual physical damage, and here it is therapeutically and clinically better to look to what is intact or reparable, to the undamaged tissues and the unimpaired and compensating functions, rather than to allow the irremediable structural damage to set a limit in advance to what may be worked for or attained.